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RESEARCH ARTICLE

FAT EMBOLISM SYNDROME: COMMON ENTITY; UNCOMMONLY DIAGNOSED

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ABSTRACT

Fat embolism syndrome is frequently a misdiagnosis. Both intensivists and orthopaedic surgeons are recognizing its more common occurrence than hitherto thought. The classical triad of hypoxia, skin involvement and neurological involvement is not always present and over dependence on scoring system may lead to misdiagnosis in our opinion. Here we report a case of Fat embolism syndrome presenting without the classical symptoms but in a critical condition. High index of suspicion lead to early diagnosis and prompt treatment with complete recovery of patient.

INTRODUCTION

Though fat embolism is a known clinical entity for more than 100 years still it is less commonly diagnosed and its pathophysiology is not completely understood. It most commonly occurs after the fracture of long bones specially the femur. As there is no definitive diagnostic test for diagnosis, various criteria have been proposed to diagnose this illness. Classical triad involving lungs skin and brain is not present all the time and a strong index of suspicion should be kept in every orthopaedic trauma to diagnose this condition. As diagnosis of fat embolism is a diagnosis of exclusion every hypoxic patient or patient having altered sensorium after orthopaedic trauma should be evaluated in a certain algorithmic way to exclude other possibilities associated with respiratory failure or neurological involvement after orthopaedic trauma. In our case report we have tried to follow the certain algorithmic approach to exclude other etiologies. Early diagnosis will help us in prompt management and better prognostication of the patient. We present the following case in accordance with the CARE reporting checklist.

CASE PRESENTATION: 23-year-old male without any comorbidities presented to us with complaints of breathlessness and uneasiness. Patient was well three days before when he slipped while walking and had severe pain in the right hip joint.

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He was diagnosed to have fracture of the right femur neck and was being operated upon in a private hospital before coming to our hospital. On examining the records, it was found that femur nailing was done. A day after the surgery he developed sudden onset of breathlessness and was referred to us for further evaluation and management. At the time of admission in emergency: GCS 15/15, BP 90/40 mm of hg, Spo2 80% on room air, heart rate 116/mt, RR 32/mt, Temperature 98.4°F. He was put on oxygen support and with 5litres of oxygen with face mask his oxygen saturation improved from 80% to 98%. A large bore cannula was put in and IV fluid resuscitation was done in view of hypotension. Patient was shifted to intensive care unit for further care. After initial stabilization of airway, breathing and circulation; he was evaluated for the cause of development of sudden onset breathlessness and hypoxemia. On auscultation biliteral lung crepitations were heard in both basal areas of the lungs. X ray chest was unremarkable. ECG showed sinus tachycardia. 2D echocardiography showed RA, RV dilatation with moderate TR, PAH and normal LV function. ABG was suggestive of hypoxia with respiratory alkalosis with lactates of 3.8 mmol/L. Complete blood count was significant for normocytic normochromic anaemia, thrombocytopenia and neutrophilic leucocytosis. His neurological status was normal and GCS was 15/15. There was no focal deficit and deep tendon reflexes were normal. He did not have any rash on his body. CT pulmonary angiogram was done to rule out pulmonary embolism, which was a strong possibility in view of sudden onset hypoxia after post orthopaedic surgery with echocardiographic findings showing RA RV dilation and TR. However, it revealed no thrombus in

the pulmonary arteries and both the pulmonary arteries were clear. Bilateral lung parenchyma showed diffused infiltrates suggestive of ARDS, his condition was critical. There was no evidence of septicaemia. Diagnosis of fat embolism was made after exclusion of all other aetiologies as there is no gold standard test for the confirmation of this diagnosis. He was given DVT prophylaxis with low molecular weight heparin while keeping a close monitoring on platelet counts. Patient was treated with supportive management and started improving after 24 hours. His tachypnoea, tachycardia and oxygen requirement improved. After 72 hours of supportive care there was remarkable improvement in his general condition with oxygen saturation maintaining on room air. He was discharged on day 6 of hospital admission.

DISCUSSION

Fat embolism is a serious complication usually occurring after lower limb long bone fracture [1]. Incidence of fat embolism is varying between < 1% to 30%. This high variability in incidence is because of lack of definitive diagnostic test along with many asymptomatic cases remaining undiagnosed [2]. Risk factors for fat embolism are usually trauma related but some non-trauma related diseases like bone tumour lysis, osteomyelitis, lipid infusion, pancreatitis are very rare causes of fat embolism [3]. To understand the pathophysiology of development of fat embolism syndrome, two theories have been proposed. One says because of increasing intramedullary pressure either because of the fracture or because of the orthopaedic procedure, fat dislodges and get enter into the venous sinuses, venous system of lungs and sometimes through the patent foramen ovale into the arterial circulation causing involvement of brain and skin [4]. Other theory is due to release of free fatty acids in the circulation leading to proinflammatory state and damage to pulmonary endothelial cells causing ARDS.

A classical triad of lung, brain and skin involvement have been described for diagnosis of fat embolism [5]. In our case this patient had only lung involvement with no evidence of any skin or brain involvement. Pulmonary manifestation is the most common involvement occurs in 96% of cases, neurological involvement occurs in 86% of cases. As classical triad was not there, we closely look for other causes of hypoxemia in such post-operative patients. Pulmonary embolism was ruled out with CT pulmonary angiogram which makes a very close differential for this entity specially in absence of a classic triad. Chest X-ray is usually normal in fat embolism as well as in pulmonary embolism. However, CT PA revealed diffuse parenchymal shadows in both lung fields which are present in fat embolism and absent in pulmonary embolism [6]. Other ancillary findings which favoured the diagnosis of fat embolism in our case were presence of anaemia, thrombocytopenia, jaundice, deranged coagulation profile along with right ventricular dysfunction[7]. As there is no gold standard diagnostic test, its diagnosis is usually made by diagnosis of exclusion along with the clinical criteria proposed by different writers like Gurd, schonfeld and lindich[8]. Gurd criteria is the most popular criteria used in diagnosis of FES. Presence of one major or four minor criteria or only two major criteria confirm the diagnosis of fat embolism. In our case one major criterion respiratory involvement with radiological changes and four minor criteria, presence of anaemia, thrombocytopenia, jaundice and presence of right ventricular dysfunction aid in diagnosis [9]. As there was no neurological symptom so we did not perform any neurological imaging in our case. Though diffuse weighted and FLAIR images of MRI of the brain shows multiple symmetrical hyperintense punctate foci in white matter looking like a star field. This finding is very sensitive for the diagnosis of fat embolism specially in the context of long bone fracture without head injury [10]. There is no specific treatment for fat embolism syndrome and is only supportive. Supportive treatment includes fluids, oxygenation and hemodynamic management [11]. Systemic corticosteroids have also been used in management of fat embolism syndrome but the literature is inconclusive [12].

DVT prophylaxis was given with low molecular weight heparin with close monitoring on the platelet count[13]. Risk benefit ratio needs to be assessed before stopping anticoagulation in patients of thrombocytopenia depending on severity of thrombocytopenia [14]. For prevention of this syndrome early surgical fixation within first 24 hour's is advised in cases of long bone fractures. In case where early fixation is not possible specially in cases of polytrauma where in initial phase a damage control surgery should be done. External fixation of fractures is preferred in comparison to nailing of the medullary cavity to reduce the risk of fat embolism in such cases [15]. Prognosis is good and favourable with good supportive care and mortality is less than 10%. Patient usually recovers completely without any permanent damage to any organ [16].

Table 1. Laboratory Parameters along with Radiological Findings

Parameters		Day of Admission	Day of Discharge
Hb (in g/dL)		7.5	8.6
TLC (in thou/mm3)		12	8.47
Platelet Count (in thou/mm3)		80,000	219
PT (in sec)		19.1	16.8
INR (in sec)		1.68	1.45
CPKMB (in U/L)		32	-
Troponin, High Sensitive (in pg/mL)		45.4	-
Sodium (in mEq/L)		136	135
Potassium (in mEq/L)		3.54	3.9
Creat. (in mg/dL)		0.81	0.73
Urea (in mg/dL)		33	35
Cl- (in mEq/L)		101	99
SGOT (in U/L)		38	23
SGPT (in U/L)		17	16
ALP (in U/L)		31	31
T. Bil (in mg/dL)		3.12	2.51
D-Dimer (in µg FEU/ml)		0.59	-
ABG	PH	7.46	-
	PCO_2	30	-
	PO_2	52	-
	HCO_3	16	-
	Lactate	3.8	-
ECG		Sinus Tachycardia	-
2 D Echo		RA, RV Dilation with moderate TR, PAH and normal LV Function.	-
X-Ray Chest		Normal	-
CTPA		Normal pulmonary arteries with bilateral diffuse pulmonary infiltrates	-

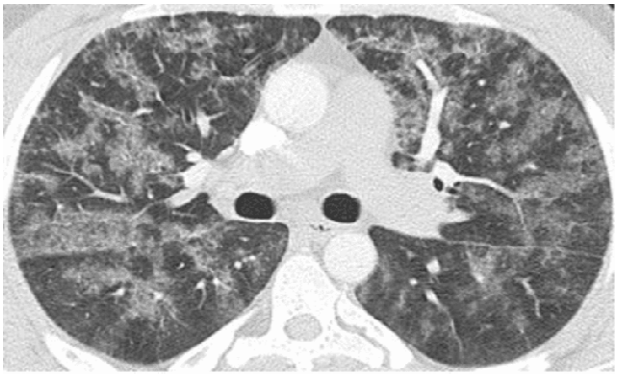

Figure 1. CT PA revealing diffuse parenchymal shadows in both lung fields

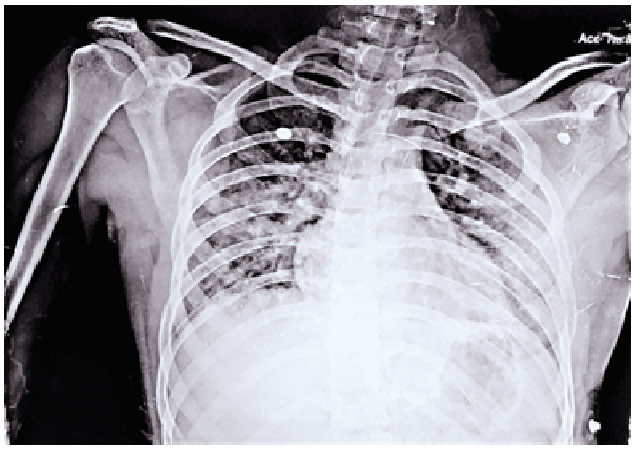

Figure 2. X-Ray Chest

Conclusion

Prevention, early anticipation and diagnosis with good supportive care is the key to success in achieving full patient recovery. Fat embolism syndrome should be suspected and evaluated in every orthopaedic trauma, presenting with hypoxemia and altered sensorium.

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